October 20-22, 1952

Dear Bruce:

Your letter of the 14th just received and duly deciphered. I appreciate your efforts to underwrite a visit to London: our plans are still very fluid, and will depend, of course, mostly on how much financial support we can accumulate here. You have, I hope, received my letter of the 4th— through some lapsus menti, I addressed it to you at the London Postgraduate Medical School, but I hope they knew well enough to forward it to you. I will assume you have received it, and go on from there.

Boyd writes, in reply, that you have seen him already to discuss the classification of Salmonella phages and their transducing action. I am not sure that his classification goes far beyond Burnet's, but it will certainly be desirable to correlate the studies. Most of the work so far has been done with PLT-7 and PLT-22, and we certainly have no right to make sweeping generalizations on this basis. Our preparation of lysates by means of heat-sterilization has probably excluded B types from previous studies. Please let me know just which ofvhis phages you will be looking at first, so that our survey here does not needlessly overlap yours. I was rather interested in his description of C20, whose host range may be rather like that of PLT-10 and one of Thompson that Spicer's working with. There will be a good deal of work to look at the other PLT's, but it may be more worthwhile to look at a better organized set, like Boyd's.

The progeny tests on SW-543 seem to bear out the linked-transduction hypothesis. I append a little pedigree outlining the results. For a time, I thought that only i's were coming through, but this was due to an inhibition of the b's by the serum agar I was using. This probably Also accounts for the possible inhibition of transduction, and may well have been due to somatic antibodies. I have some better (absorbed) sera now that should give simpler results. The transductions from other serotypes (gm, gp, r, enx and possibly c) have been checked, very satisfactorily. One appears to get b's and the FA type in each case, so far apparently monophasic. I think it most important, however, to do a thorough serological analysis both on the B's and the i's-- there are some hints of partial b's in the following: a) the inhibition by i sera, sans positive agglutination, which may have been due to somatic antibody, or possibly to some common components, and b) the occurrence of b phases (especially in SW-543 or H901 x-abony FA which swarm through b agar to give a phase that reacts only more weakly with the b reagent. I will send representatives of these. I have not been able to find a second phase in SW-609 except the inagglutinable "j"?? previously mentioned. The i transinductions seem equally unstable. Be careful with SW-435 as a recipient strain for antigen studies: its motility is rather erratic. I have a SW-435 x- abony set that I intended to use for phase variation study, but mustn't trust the results (which are of a negative sort). SW-541 is self-plaquing. Norton is ready to admit that SW-572 is a fluke. It involved heated FA as a control, and the inactivation may have been imperfect.

An unstable transinduction has been picked up from SW-666 x-PLT22 on EMB Gal. It looks just like a Gal+/- heterozygote in E. coli. Unlike the unstable transductions by lambda, this one gives stable + and stable-. Thus it would correspond more closely to the track-initials of swarms. Perhaps the frequency of these types is

higher than we can easily detect, but I have found only this one (SW-684) in several dozen tests. FA(684) seems to transduce only typical Gal+. I have gotten several swarms from SW-684 x- PLT22, but all were pure Gal+ or -. In view of the instability of 684, it is hard to tell whether the negative result is significant. In broth cultures, one gets perhaps as much as twice as many Gal- as Gal+ "segregants", but it is impossible to say what this means. At any rate, we have a reasonable analogy to the tracks.

SW-665 (541 Xyl-) is behaving very strangely on xylose. The transductions give inconsistent reactions, possible a temperature effect that needs to be traced. SW-541 itself is relatively weak on xylose, but does not give quite the same erratic scoring.

No linkage of motility to Xyl or Gal transductions has been found with either strain.

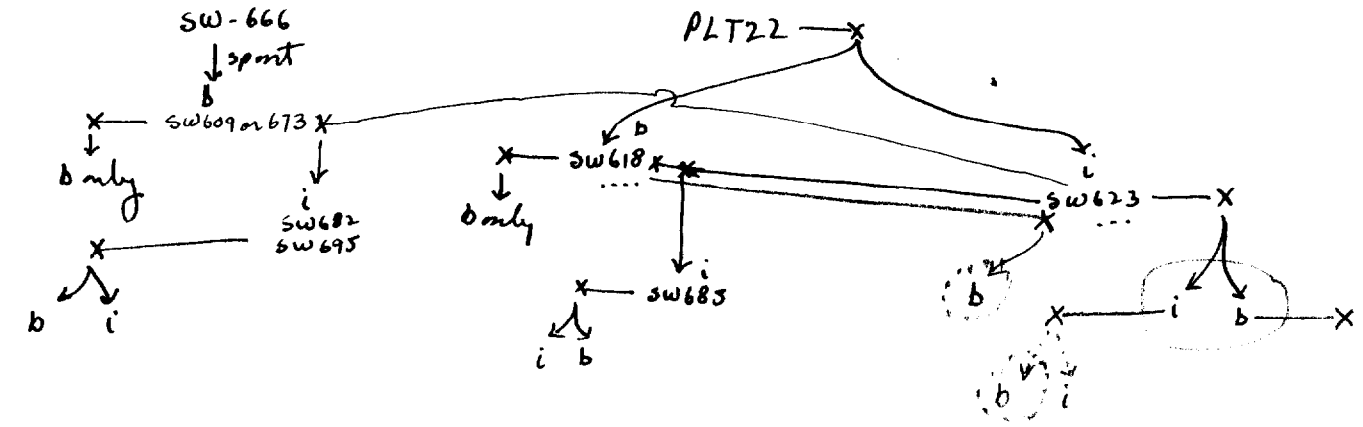
To save time, I am recording Edwards' serotypes as SW-703-900, corresponding to his numbers 1, etc.... in the Ky.Ag. Bull. 50 SW-703 (in case you took that along) has a non-paratyphi B Salmonella in it, presumably a contaminant, not yet typed. The purified SW-703 is a typical b--1,2.

What else needs to be done to clean up the summer's work? It shows signs already of branching out over the whole field, and we should call a halt somewhere soon. Are you working with pneumococcus as well?

Miss Mislap, at Basel, sent the rather uninteresting history of SW-543. It was isolated from a culture from an insane asylum habitant in 1941. He excreted para B for about a year, was finally recorded as negative. ~~[illegible]~~ His clinical diagnosis was Paratyphoid fever, hardly any details. An "Ohrekzem" is mentioned, no obvious connection. They lost track of him in 1945. Cultures were not saved. Presumably a motile paraB was sent to Kauffmann, and he isolated SW-543 from this, but there is no clear statement on this except from Kauffmann. The case was isolated.

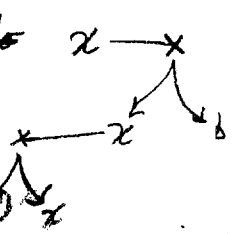
If no backcross parent is given, assume SW543 or SW666.

→ x transduced to.
← transduced by.



— done
— in progress
inconsistent with the alternative to linkage hypothesis.

I am also ~~planning to~~ initiating



(685 → and 682 →)

for x = phase II of typhimurium

x = r or some other.

The two-step transductions now seem superfluous experiments. They were predicated on the non-linkage hypothesis. 682 → was to test which locus ("H" or "X") of SW 543 was spontaneously mutable.

Sincerely, Josh.